a growth obtained. No bacteria were found in the smears made directly from the excised and crushed gas-containing tissue.

SUMMARY. 1. Those cases of influenza-pneumonia at Camp Dix which showed gas in various fasciæ showed clinically no evidence of an intercurrent infection.

2. Three gas cases ran an uneventful course and recovered without surgical procedure.

3. At autopsy the gas-containing tissue showed little blood in the vessels. It presented no discoloration, no congestion, no induration or other signs of inflammation.

4. Bacteria were not recovered.

5. Gas was not found within the muscle sheaths.

6. In the gas cases coming to autopsy the air-containing alveolæ appeared emphysematous in the more nearly consolidated lung lobes.

7. The distribution of the gas corresponds with what we would expect to be the dissemination of gas escaping from the weakened alveolæ of the lung under pressure.

8. We are of the opinion that the "gas" in the above cases of pneumonia is air from the lung which reaches its distribution as a result of purely mechanical factors. We do not believe the gas is of bacterial origin.

RENAL DIABETES.[1]

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THAT diabetes is not always a rapidly fatal disease has long been recognized by medical writers, the diagnosis being founded upon the chief sign of the disease, the persistent presence of sugar in the urine. In many cases this is the only symptom, the patients enjoy excellent health, the glycosuria persists and seems to be little influenced by diet, and, after years under observation, may remain unchanged or may entirely disappear. The association of the kidneys with the etiology of diabetes is not to be wondered at, especially from the 17th century, when Thomas Willis discovered that the pathological change in the urine was the presence of sugar, and Dobson, in 1775, demonstrated that the blood contained sugar before it appeared in the urine. One naturally conceived of a permeability point in the concentration of blood sugar at which the kidney tolerance would be overcome and sugar would escape into the urine. In the ordinary cases of diabetes it was observed that the urine sugar varied more or less directly with the diet and the patient's tolerance was estimated by the amount of carbohydrate that could be ingested without producing glycosuria. The question arose whether one was dealing with a condition in which this permeability point was lowered by organic or functional kidney changes or whether the kidneys were unaffected and the symptoms due to a constitutional defect in carbohydrate metabolism. Early writers, as Galen, Aetius of Amida and others, considered it to be a kidney disease. Theophrastus Patrocelsus, however, described it as a constitutional disease showing itself in marked pathological changes in the urine. In 1878 Claude Bernard,[2] by very imperfect methods, demonstrated that an excess of sugar in the blood was the direct cause of glycosuria.

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[1] Read in part before the Society for Experimental Biology and Medicine, May 17, 1916, Proceedings, xiii, 153.

Von Mering's[3] demonstration of phlorizin glycosuria in 1886 raised the question as to whether a like condition occurred clinically in man, a state in which the kidney permeability point to sugar was so lowered that glucose appeared in the urine in the presence of a subnormal or normal blood sugar. In 1896, Klemperer[4] reported a case which he styled "Renal Diabetes" and stipulated that for such a diagnosis one must have the following postulates fulfilled:

I. That the glycosuria should be within wide limits independent of the amount of carbohydrate in the food.

II. That the sugar in the blood should at least not be increased but should rather be diminished.

III. That the development of a nephritis decreases the glycosuria.

Schupfer,[5] in criticism, pointed out the fact that although a complicating nephritis usually causes a lessening or cessation of a mild glycosuria, in other cases no change can be found and claimed this to be the case in arteriosclerosis complicating diabetes. Lüthje[6] in 1901 claimed that in order to make the diagnosis one must ascertain that the glycosuria develops as a direct result of a nephritis, that the blood sugar be below normal and that no relation exists between the blood sugar and the extent of the glycosuria. In 1908 Bonniger[7] questioned the diagnosis made in all previous cases as the blood sugar estimations were based upon the reducing power of whole blood. This, he said, gave us erroneous values as he was under the impression that blood corpuscles contained very little sugar. Tachau[8] further decorated the diagnostic requisites by stipulating that the glycosuria, in addition to being completely or in a great degree independent of the diet, must persist upon a diet free from carbohydrates and that it be not greatly increased, following the ingestion of large amounts of sugar.

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[2] Leçons sur le diabète, Paris, 1878, 75.

[3] Verhandl. d. Cong. inn. Med., Wiesbaden, 1886, v, 185.

[4] Verein f. innere Med., Berlin, 18 Mai, 1896.

[5] Il Policlinico, 1900, vii-m, 1.

[6] München. med. Wchnschr., 1901, xlviii, 1471.

[7] Deutsch. med. Wchnschr., 1908, xxxiv, 780. Verhand. d. Cong. f. inn. Med., 1913, xxx, 178.

[8] Deutsch. Arch. f. klin. Med., 1911, civ, 448.

Von Noorden[9] claimed that blood sugar estimations must be the deciding factor, and in this connection, Frank,[10] in 1910, again brought up Bonniger's criticism of whole-blood estimations. Jacobsen,[11] in 1913, published some very interesting data on alimentary hyperglycemia and glycosuria. He found that one hour following the administration of 100 grams of glucose the hyperglycemia had passed off in most of the cases examined and the blood sugar test at this time showed only a very slight increase. He pointed out that this is probably what happened in the cases diagnosed as renal diabetes.

De Langer,[12] in 1914, stated that to make a diagnosis of renal diabetes the glycosuria need not be uninfluenced by the diet and pointed out that in phlorizin diabetes the urine sugar is greatly influenced by ingested carbohydrates.

Galambos,[13] in the same year, differentiated between mild diabetes mellitus and renal diabetes as follows: "When a chronic glycosuria is present with but a questionable or no hyperglycemia, we have to do with either a diabetes mellitus with increased kidney permeability or with renal diabetes. When the blood sugar is slightly increased the case is one of diabetes mellitus with increased renal permeability. When, however, the blood sugar is subnormal or normal, or when alimentary hyperglycemia is absent, the case is one of renal diabetes. "Glycosuria largely independent of the diet speaks for renal diabetes."

Salomon,[14] also in 1914, cited the work of Porges, Leindorfen and Stresower and Frank as showing that an independent glycosuria with low blood sugar values can exist in patients showing no sign of kidney disease. He stated that renal diabetics excrete small amounts of sugar daily, 10 to 12 grams, and that this is uninfluenced by diet; also that 100 grams of glucose in the morning do not increase the sugar excretion.

The consensus of opinion seems to have been that renal diabetes was a condition in which the patient had none of the classical symptoms of diabetes mellitus but did have a low-grade glycosuria, little influenced by diet, and which was present at a time when the blood sugar was normal. In view of the fact that Lewis and Mosenthal[15] have but recently given an excellent review of the cases so far reported a repetition would seem out of place at this time.

[9] Die Zuckerkrankheit., Berlin, 1912, 527.

[10] Arch. f. exper. Pathol. u. Pharmakol., 1913, lxxii, 387.

[11] Biochem. Ztschr., 1913, lvi, 471.

[12] Berlin klin. Wchnschr., 1914, li, 1792.

[13] Deutsch. med. Wchnschr., 1914, xl, 1301.

[14] Ibid., xl, 217.

[15] Johns Hopkins Hosp. Bull., 1916, xxvii, 133.

In reviewing the cases, however, one is impressed by the fact that most of the diagnoses were based upon the observations that the patients had no diabetic symptoms and that the glycosuria seemed to bear no relation to the diet. When it came to actually determining the point of permeability to sugar and whether or not sugar was escaping from the kidney at the very time when the blood sugar was normal, little can be found to establish the diagnosis.

The case described by Gram[16] had a low glycosuria of from 0.4 to 0.6 per cent. at the time when synchronous specimens of blood contained 0.085 to 0.1 per cent. sugar. Lewis and Mosenthal[14] reported a case with a constant glycosuria in twenty-four-hour specimens, during a period of five days' starvation; blood tests made on the third and fifth days gave normal values (0.11 per cent.). The patient had no diabetic symptoms. We cannot draw conclusions from their synchronous blood and two-hour urine tests. The blood specimens were taken at a time when alimentary hyperglycemia had passed off, but the urine at that time would still normally have sugar as a result of the hyperglycemia.[17] Other blood specimens were taken just before eating and the synchronous urine specimens contained the alimentary glycosuria of from one to two hours.

Murlin and Niles[18] have described a case which they consider to be one of renal glycosuria. The patient, a man, aged twenty years, suffered from loss of weight, furuncles, polyuria, increased thirst and weakness. The daily excretion of glucose varied from 14.7 to 26.9 grams and bore no relation to the carbohydrate content of the diet, which varied from 14.9 to 100 grams. The blood-sugar estimations were made five hours after breakfast and gave normal values on several days. One day's starvation rendered the urine sugar-free. No synchronous blood and urine tests were made and nothing is known of the permeability point to sugar or of the nature or extent of alimentary hyperglycemia and glycosuria. The blood sugar findings are such as one would expect in mild diabetes.

The above case reported by Gram is certainly one of "glycosuria with normal glycemia," as Allen states it. Several other cases, including that of Lewis and Mosenthal and that of Bonniger, seem to approach this type, but the data from which to draw conclusions are insufficient. It is known that the blood sugar level when uninfluenced by diet or by glycogenolysis varies in different individuals, high levels being found in diabetes of long standing and in nephritis. So it would be quite possible for a nephritic whose blood sugar level is above 0.2 per cent. to have a glycosuria of renal origin which would not be dependent upon an increase of blood sugar in that individual. One of the cases about to be reported is of this type.

The value of the blood sugar method has frequently been brought into question in deciding the type of the case. Bonniger's and Frank's criticism of whole blood estimations is not without foundation, as the sugar concentration of the plasma alone must be the deciding factor influencing the kidney cells. However, in the light of recent work,[19] this would tend to include more cases as renal glycosuria, since the corpuscles seem to have, as a rule, a slightly greater concentration of sugar than the plasma.

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[16] Hospitalstidende, Copenhagen, 1915, lviii, 329.

[17] Frank: Ztschr. f. physiol. Chem., 1910, lxx, s. 129.

[18] Am. Jour. Sc., 1917, cliii, 79.

The following cases are reported as showing two distinctly different types of glycosuria which seem to be of renal origin. The blood sugar estimations were made by a modification of the Lewis and Benedict method already described.[20]

CASE I.—Mrs. M. B., an American housewife, aged thirty-one years, was admitted to the service of Dr. Quintard in the New York Post-Graduate Hospital on July 30, 1915. Case History No. 5356. She was sent to the hospital on account of an intractable glycosuria complicating pregnancy.

Family History. Mother died of "inflammation of the bowels." Father, aged seventy-four years, living and in good health. Two brothers and four sisters are living and in good health. Her family have all been of an extremely nervous temperament. No history of diabetes, obesity, gout, goitre, tuberculosis, gigantism or disorders of the kidneys, stomach or heart.

Personal History. Born in Westminster, Vermont, of old New England stock. She experienced poor health from early infancy and has always been poorly nourished. Her mother told her that when a child the urine was irritating, and that only by the best of care could the vulva be kept in a healthy condition. From early childhood she has had a weak stomach and suffered from severe attacks of indigestion. These attacks were usually accompanied by marked urticaria, especially so following the ingestion of fish. She has a peculiar susceptibility to the sting of the honey-bee. On one occasion, four years ago, she almost died from the effects of a sting. It was followed by nausea, vomiting, purging, loss of consciousness, marked edema and extreme weakness of pulse. Brisk stimulation brought about recovery in a few hours. Had chickenpox when a child; catarrhal jaundice at the age of thirteen; normal menstruation began at fourteen; mumps when eighteen. Married at the age of twenty-three and two months later had a second attack of catarrhal jaundice. Her first child was born one and a half years after marriage. Early in the pregnancy she noticed burning, frequency and urgency of urination. Her physician found sugar in her urine and made a diagnoss of diabetes mellitus. Before this date (1908) her urine had never been tested for sugar.

History of Present Illness. Following the discovery of the glycosuria she was placed upon a restricted diet, but as this was followed by a rapid loss of weight and extreme weakness it was soon abandoned. The patient returned to her usual diet and quickly regained both weight and strength. After two weeks the urine was again examined for sugar, which was present to the extent of 5 per cent., or about the same amount as before the restricted dieting. This glycosuria persisted throughout the pregnancy, which was otherwise uneventful. Her diet continued unrestricted, her weight increased to 150 pounds and she looked and felt in perfect condition. A baby girl, weighing ten and a half pounds, was delivered under ether anesthesia. An endometritis lasting three weeks followed the birth of this child. Recovery was good, and since that time the patient has been in fair health.

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[19] C. V. Bailey: Proc. Soc. Exper. Biol. and Med., 1916, xiii, 153.

[20] Myers and Bailey: Jour. Biol. Chem., 1916, xxix, 147.

Four years ago another unsuccessful attempt was made to render her urine sugar-free, but was quickly abandoned, due to rapid loss of weight and extreme weakness. Since that time her diet has been unrestricted. Her appetite has always been good—never abnormal. She has not had increased thirst. The urine volume has been about 1500 c.c. She has had a distaste for sweet things, but is fond of fruit. She has always been undernourished, nervous and rather easily exhausted, but has been able to do all the housework for three adults and a child. In the past seven years there have been no subjective urinary symptoms. Her chief complaint has been of occasional attacks of indigestion, when she would suffer from headache, nausea, vomiting and epigastric distress. No hematemesis. Bowels regular. No constipation. Diarrhea at times. No blood or excess of fat in the stools. Menstruated regularly up to three months ago.

Physical Examination. Poorly nourished, symmetrical adult female of rather querulous temperament. The skin is very transparent; cheeks flushed; lips very red; veins of neck, chest and temples prominent; eyelashes long; slight seborrhea of scalp; pupils equal, regular and react well to light and accommodation; no exophthalmos; no widening of palpebral fissures; von Graefe's sign negative; corneal sensibility slightly decreased; upper and lower teeth missing; mucous membrane bright red; throat apparently normal; thyroid gland slightly enlarged, symmetrical and does not pulsate; no tremor of extended fingers; reflexes all present and active; no swaying on standing with feet together; no ataxia; chest symmetrical, subcostal angle acute, expansion equal and good; breasts enlarged, slightly tender and areolæ pigmented; examination of lungs reveals nothing abnormal; cardiac dulness extends to right sternal border; apex beat palpable in fifth intercostal space in midclavicular line; heart sounds normal—no adventitious sounds; arteries barely palpable; pulse 84, regular and equal; blood-pressure: systolic 110, diastolic 65; respirations 22; temperature normal; abdomen enteroptotic and flatulent; liver palpable at costal margin; upper pole of right kidney can be palpated; spleen not enlarged; bimanual examination reveals an enlarged uterus;

no abdominal tenderness or abnormal masses are present; no edema of extremities; hemoglobin 68 per cent. (Sahli); red blood cells 4,444,000; leukocytes 7900, of which 64 per cent. are polymorphonuclear; urine light straw colored—specific gravity 1047—and shows a slight trace of protein, acetone and diacetic acid, and sugar 5 per cent.; microscopically—a few hyaline and granular casts; phenolsulphonephthalein excretion on August 10, 1915, was 39 per cent. in two hours; on August 17 the excretion was 42 per cent. in two hours.

On August 3, 1915, her blood in the morning before breakfast showed 0.09 per cent. sugar, and the urine at the same time contained 1 per cent. sugar. A comparison of the blood and synchronous urine sugar is found in Table I, Case I. The effect of starvation upon her weight and excretion of sugar is shown in Table II, Case I, the patient at the same time being at rest in bed.

TABLE I.—CASE I.

Date.	Blood sugar. Per cent.	Synchronous urine sugar. Per cent.
Aug. 3, 1915	0.089	1.00
Aug. 5, 1915	0.110	1.60
Aug. 6, 1915	0.130	2.10
Aug. 11, 1915	0.098	3.10
Aug. 25, 1915	0.092	1.25
Oct. 13, 1916	0.116	8.40

This table simply shows that with normal blood-sugar values, sugar was constantly excreted by the kidneys. As the rate of excretion of urine is not known the relationship of sugar excretion to blood-sugar concentration cannot be expressed.

TABLE II.—CASE I.

Date.	Diet.	Weight. Pounds.	Glucose excreted. Grams in twenty-four hours.
Aug. 3, 1915	Regular	103	35.0
Aug. 5, 1915	Hunger	99	18.3
Aug. 6, 1915	"	96	14.7
Aug. 7, 1915	"	95	19.5

Test of Alimentary Hyperglycemia and Glycosuria. On the morning of August 11, 1915, synchronous blood and urine specimens were obtained, the patient being in the fasting state. She was then given 30 grams of glucose in 225 c.c. weak tea. Blood and urine specimens were obtained at frequent intervals for a period of six hours. The results are tabulated in Table III, Case I, and in Chart I, Case I.

It will be seen that the patient excreted glucose at the rate of 1.29 grams per hour when the blood contained 0.098 per cent. sugar. Alimentary hyperglycemia was slightly delayed and prolonged, as is found in mild cases of nephritis. Urinary excretion greatly increased on the subsidence of the hyperglycemia.

The greatly increased permeability of the kidneys to sugar is well shown at the one and a half hour period, when the rate of glucose excretion was 6.7 grams per hour while the blood sugar was at a point where normal kidneys just become permeable.

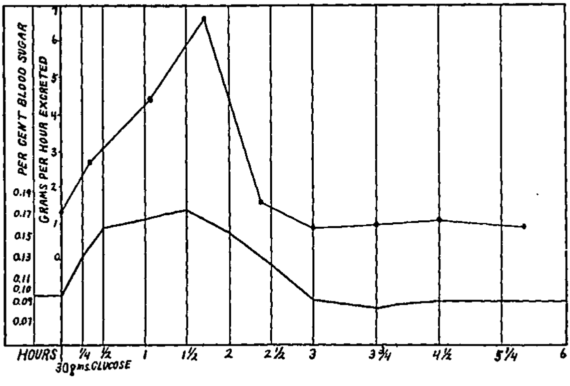
TABLE III.—CASE I.

M. B., August 11, 1915.

Time.		Blood sugar. Per cent.	Urine.				Fluid intake. c.c.
			c.c. per hour.	Chlorides. Grams per hour.	Sugar.		
A.M.					Per cent.	Grams per hour.	
9.15	....	0.098	60.0	0.0460	3.120	2.000	
9.45	....	30 grams glucose.	44.0	......	2.940	1.290	225
10.00	¼ hr.	0.135					
10.20	½ hr.	0.159	58.0	0.1056	4.830	2.830	225
10.30							
10.45	1 hr.	0.168	.....	......	......	......	225
11.00	1¼ hrs.	0.172	66.0	0.1450	6.660	4.398	
11.10							
11.15	1½ hrs.	0.176					
11.45	2 hrs.	0.156	97.0	0.2180	6.750	6.700	225
12.15	2½ hrs.	0.105					
12.25	....	......	159.0	0.2540	1.040	1.650	
12.45	3 hrs.	0.093					
1 P.M.	....	......	94.0	0.1690	0.952	0.897	
1.25	3½ hrs.	0.086					
1.45	4 hrs.	0.088	150.0	0.2100	0.657	0.986	225
2.00							
2.15	4½ hrs.	0.092					
3.00	5¼ hrs.	0.092	90.0	0.1400	1.219	1.097	
3.45	6 hrs.	0.092	84.0	0.1340	1.086	0.912	225

M.B. 11-VIII-15

CHART I. CASE I.



The patient was put upon a mixed diet containing about 50 grams of available carbohydrate. Upon this diet her twenty-four-hour urine contained not more than 2 per cent. glucose while her normal weight was sustained. Her freedom from unpleasant symptoms, however, prompted her to return to unrestricted diet upon leaving the hospital on August 25. In two weeks her weight increased twenty pounds. The second pregnancy was uneventful, and on January 28, 1916, she was delivered of a healthy baby weighing nine pounds, nine ounces. Glucose was constantly present in the urine.

In October, 1916, nine months after the birth of her second child, further tests in alimentary hyperglycemia and glycosuria were carried out. At this time the percentage glucose in her daily urine had greatly increased, being 7.4 per cent. on October 15, 6.94 per cent. on October 16, and 9.2 per cent. on October 17. On the morning of October 13, synchronous blood and urine specimens were obtained, the patient being in the fasting state. She was then given 30 grams of glucose in 400 c.c. of weak tea. Blood and urine specimens were obtained at frequent intervals for the succeeding six hours. The results are shown in Table IV, Case I, and Chart II, Case I.

The initial blood sugar value was within normal limits, but at the time sugar was being excreted by the kidneys at the rate of 2.98 grams per hour.

The blood sugar curve reached its highest point in one hour and returned to its original value in two and a half hours, as is normally the case. The rate of sugar excretion increased with the blood sugar, so that when the latter was at a concentration of 0.189 per cent., sugar was being excreted at the enormous rate of 9.9 grams per hour. (A comparison of the rate of fluid excretion with the concentration of sugar in the blood is interesting, as during the rapidly developing hyperglycemia the fluid excretion dropped off markedly and then quickly increased as the kidneys rapidly removed the excess of sugar from the blood. This decreased excretion occurred in spite of the fact that 400 c.c. of fluid were imbibed with the glucose.)

If we compare the findings at this time with those of the similar test taken fourteen months previous (at which time the patient was four months pregnant) we notice that the blood sugar in the fasting state has increased to the normal value. This, however, is easily explained, as the tests in August, 1915, were made following three days of fasting, while the latter tests followed but a ten-hour fast. The excretory power of the kidneys for glucose has increased, and the more rapid rise of blood sugar following the ingestion of glucose probably indicates an increase in absorptive power of the intestines.

The general condition of the patient was unchanged. Her chief complaint was lack of reserve energy. This was most distressing upon restricting the diet. The urine sugar remained above 6 per cent. in the twenty-four-hour specimens.

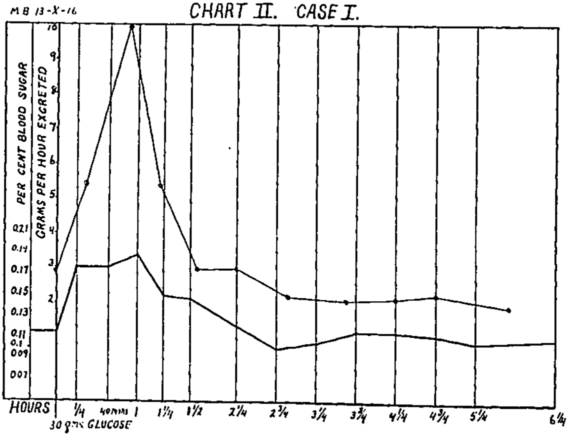
The patient was again examined on December 20, 1917. Her physical condition remained unchanged. She stated that for the preceding fourteen months her health had been excellent, her only complaint being lack of reserve energy. Her diet was unrestricted. Examination of a twenty-four-hour urine specimen on November 4, 1918, showed an excretion of 1440 c.c.; specific gravity 1040; sugar 4.5 per cent.; albumin negative.

TABLE IV.—CASE I.

M. B., October 13, 1916.

Time.		Blood.		Urine.			Fluid intake. c.c.
					Sugar.		
A.M.		Hemoglobin. Units.	Sugar. Per cent.	c.c. per hour.	Per cent.	Grams per hour.	
9.15	....	67	0.116	63.0	8.3	2.98	
		30 grams glucose.					
9.30	....		......	......	......	......	400
9.45	¼ hr.	62	0.177				
10.10	⅔ hr.	61	0.177	36.0	11.9	5.35	
10.30	1 hr.	62	0.189				
10.40	....	......	......	45.0	3.0	9.90	
10.50	1⅓ hrs.	63	0.150				
11.00	....	......	......	33.0	1.6	5.28	
11.10	1⅔ hrs.	64	0.147				
11.30	....	......	......	80.0	3.7	2.96	
11.45	2¼ hrs.	65	0.120	120.0	2.5	3.00	
12 M.							
12.15	2¾ hrs.	65	0.100	37.0	6.2	2.31	
12.45	3¼ hrs.	66	0.106				
1.15	3¾ hrs.	67	0.116				
1.30	....	......	......	56.0	5.6	2.09	
1.45	4¼ hrs.	67	0.114				
2 P.M.	....	......	......	36.0	6.0	2.16	
2.15	4¾ hrs.	67	0.112				
2.30	....	......	......	31.0	6.5	2.21	
2.45	5¼ hrs.	68	0.106				
3.30	....	......	......	28.0	6.8	1.90	
3.45	6¼ hrs.	68	0.108				

CHART II. CASE I.



SUMMARY. The case reported is one of constant glycosuria without hyperglycemia. Alimentary hyperglycemia while the patient was pregnant was slightly delayed and prolonged, this being probably due to decreased functional activity of the bowels and kidneys. Alimentary hyperglycemia in the non-pregnant state was of the normal type,[18] the curve reaching its highest point in about one hour, returning to the original value in two and a half hours, falling to a lower point in three hours, then increasing and maintaining its level in three and a half to four hours' time. Alimentary glycosuria was excessive. It varied directly with the glycemia and was present to a marked degree even in the presence of a hypoglycemia, the permeable point of the kidneys being below a blood sugar concentration of 0.086 per cent. (the normal kidneys being permeable between 0.16 and 0.17 per cent.). The patient's history points strongly to this being a congenital condition.

Since the accidental discovery of sugar in her urine in 1908 her condition has remained unchanged, although her diet has never been restricted. Her daily output of sugar is about 20 grams.

CASE II.—J. E. P., English-American, aged sixty-two years, a toolmaker by trade, was admitted to the New York Post-Graduate Hospital, to the service of Dr. Quintard, on August 25, 1915. Case History No. 5940. He complained of swelling of the feet and abdomen and shortness of breath.

Family History. Mother died at seventy-six, cause unknown; father died at thirty-nine, cause unknown. Three brothers and three sisters are alive and well. No history of obesity, diabetes, nephritis, gout, goitre, tuberculosis, rheumatism or heart disease.

Personal History. Born in England of English parentage. Came to the United States when a young man. With the exception of a gall-bladder disturbance, for which he was operated upon thirteen years ago, he has enjoyed good health up to the onset of his present illness. He has no knowledge of disease in childhood and denies having had venereal disease. He was married twice; the first wife bore eleven healthy children and no miscarriages; the second wife is alive and in good health.

Habits. Alcohol in moderation. For years he has used tobacco to excess and has been a heavy meat-eater. He has never engaged in dangerous occupations.

History of Present Illness. Dates back two years, when he noticed dizziness, spots before the eyes, failing vision and severe headaches. Six months later he noticed a puffiness under the eyes and swelling of the feet and legs. These symptoms gradually increased in severity and three months ago he developed dyspnea on slight exertion, and the necessity of urinating at least once at night. Shortly before coming to the hospital he became very restless and orthopneic. His appetite has always been good, never ravenous; bowels regular; never jaundiced; no blood in stools; no nausea or vomiting; no gastric disturbances, cough or hemoptysis. His daily output of urine has not been increased. He has had none of the symptoms associated with diabetes (polyphagia, polydypsia, polyuria, obesity, emaciation, skin lesions, pruritus or neuritides); no symptoms of disturbance of the hypophysis, thyroid, pancreas or adrenals. He has no knowledge of having passed sugar in his urine.

Physical Examination. Well-developed and well-nourished male. Height 5 feet 6 inches; weight 198½ pounds, lying in dorsal position moderately dyspneic; marked generalized edema, especially of feet, legs, thighs, scrotum and hands; abdomen pendulous and edematous; slight cyanosis of ears, nose and lips; skin dry, scaly and transparent; soft edema about eyes; sclera clear, pupils equal, regular in outline and react well to light and accommodation; von Graefe's sign absent; no exophthalmos; no widening of palpable fissure; tongue coated; teeth decayed; marked pyorrhea; nothing abnormal found in nose or throat; veins of the neck engorged; thyroid palpable but not enlarged or pulsating; thorax emphysematous and symmetrical; expansion poor; respirations shallow; moist rales heard over base of lungs and free fluid can be demonstrated in the pleural cavities; temperature 98°; pulse 84; respirations 22; pulse irregular and unequal; arteries hypertrophied; apex beat diffuse and heaving; no thrill over precordium; area of cardiac dulness 3 cm. to right and 12 cm. to left of midsternal line; aortic second sound accentuated; no adventitious sounds; blood-pressure: systolic 190; diastolic 130; marked edema of abdominal walls; old scar in region of gall-bladder; moderate ascites; a small indirect inguinal hernia on right side just emerges from the external ring but does not descend into the scrotum; on the left side is a small hernia just reaching the external ring; scrotum very edematous and the tunica vaginalis on the right side distended with fluid; liver, kidneys and spleen not palpable; reflexes (abdominal cremasteric, knee-jerk, ankle-jerk, etc.) all present and active; no ataxia; Rhomberg negative; skin shows a dark brown pigmentation over the lower chest and the upper abdomen; dry, scaly eczema over both tibiæ.

The fundus of the right eye shows an angiopathic neuroretinitis, with numerous areas of fatty degeneration in macular region and numerous flame-shaped hemorrhages along the vessels near the disks. The left eye shows a cloudy vitreous with large floating bodies occluding the view of the fundus.

Volume of twenty-four-hour specimen of urine 625 c.c.; color dark amber; specific gravity 1025; dry protein per liter 3.1 grams; sugar 0.6 per cent.; total chlorines as NaCl 6.6. grams; acetone negative; diacetic acid negative; many hyaline and granular casts; phenolsulphonephthalein excreted 8 per cent. in two hours.

An examination of the blood shows 83 per cent. hemoglobin; 4,592,000 erythrocytes; 12,400 leukocytes, of which 74 per cent. are polymorphonuclears and 23 per cent. lymphocytes.

Complement-fixation test for syphilis negative. Chemical blood analysis during fasting showed blood sugar 0.18 per cent.; chlorides as NaCl 0.73 per cent.; urea nitrogen 23 mg. per 100 c.c. blood; uric acid 4.0 mg. per 100 c.c. blood; creatin 7.4 mg. per 100 c.c. blood; creatinin 1.9 mg. per 100 c.c. blood.

Subsequent History. The patient remained in the hospital ten months and underwent ordinary treatment for chronic parenchymatous nephritis. At times he showed marked improvement, followed by relapses, and finally died on June 1, 1916. During his stay in the hospital his urine was never free from sugar, the percentage remaining in the neighborhood of 0.5 per cent., varying slightly inversely with the volume and seemingly independent of the diet.

Test of Alimentary Hyperglycemia and Glycosuria. On the morning of December 13, 1915, before the patient had anything to eat or drink, specimens of his blood and urine were obtained. He was then given 33 grams glucose in 250 c.c. of weak tea, and specimens of blood and urine were obtained as frequently as possible for the succeeding five and a quarter hours. The findings are recorded in Table V, Case II, and Chart I, Case II.

It will be noticed that the initial blood sugar value is that commonly met with in cases of severe nephritis.[21] The urine, however, contained 0.52 per cent. glucose, the excretion being at the rate of 0.22 gram per hour. Alimentary hyperglycemia was delayed, reaching its maximum in one and a half hours and not returning to normal until four and a quarter hours had elapsed. The rate of urinary excretion decreased and was uninfluenced by the fluid intake. The rate of sugar excretion varied directly with the excretion of urine and bore no relation to the concentration of sugar in the blood. The blood volume, as indicated by the hemoglobin, showed a slight increase at the onset of the hyperglycemia, but returned to the original value in one and a half hours.

On March 11, 1916, the test was repeated, using on this occasion 75 grams glucose in 450 c.c. of weak tea. The general condition of the patient was the same as at the time of the previous test. The results are shown in Table II and Chart II, Case II.

One sees that in this second test the alimentary hyperglycemia was delayed and prolonged. The greatest concentration occurred at the end of two hours while his normal value had not been reached at the end of six and a quarter hours.

The rate of urinary excretion decreased during the development of the hyperglycemia and was practically uninfluenced by the fluid intake. The rate of sugar excretion again fell rapidly with the development of the hyperglycemia and had not begun to increase by the end of the test.

If one were to judge this case by a consideration of the concentration of sugar in the urine it would seem that we had a patient who excreted urine containing about 0.5 per cent. glucose and that this was independent of the blood sugar up to the latter's concentration of about 0.26 per cent. At this point the kidneys became intolerant and more sugar appeared in the urine.

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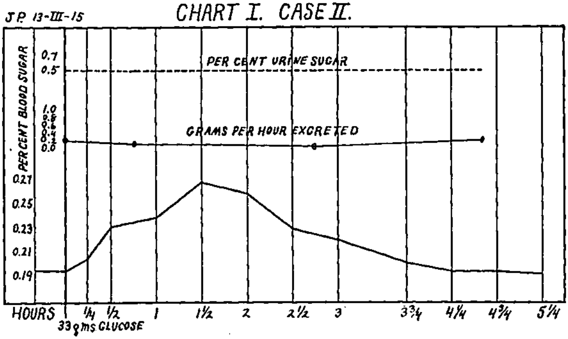
[21] C. V. Bailey: Proc. Soc. Exper. Biol. and Med., 1916, xiii, 153.

TABLE V.—CASE II.

J. E. P., December 13, 1915.

Time.		Blood.		Urine.					
					Sugar.				
A.M.		Hemoglobin. Units.	Sugar. Per cent.	c.c. per hour.	Per cent.	Grams per hour.	Specific gravity.	Chlorides, grams per hour.	Fluid intake. c.c.
9.25	....	67.5	0.196	42	0.52	0.220	1015	0.193	250
9.30		33 grams glucose.							
9.45	¼ hr.	64.0	0.208						
10.00	½ hr.	65.0	0.234						
10.30	1 hr.	65.5	0.240	36	0.51	0.186	1015	0.123	250
11.00	1½ hrs.	67.5	0.270						
11.30	2 hrs.	67.0	0.260						
12 M.	2½ hrs.	68.0	0.232						
12.30	3 hrs.	67.5	0.224	33	0.51	0.170	1016	0.142	
1.15	3¾ hrs.	67.5	0.204						
1.30									
1.45	4¼ hrs.	67.5	0.196						
2.15	4¾ hrs.	68.0	0.196	42	0.52	0.210	1013	0.190	
2.45	5¼ hrs.	67.5	0.192						

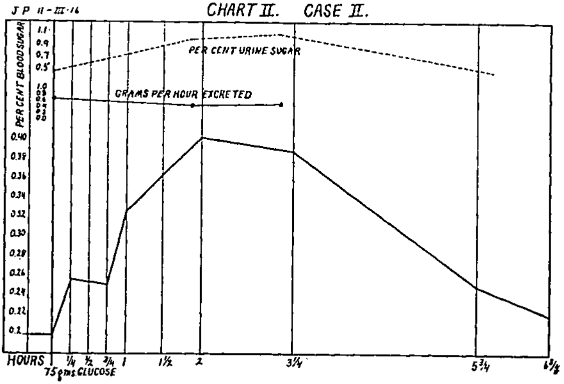


However, we see that the reverse is the case when we consider the actual excretory work done by the kidneys, the glucose being expressed in grams per hour. In Table I the kidneys excreted 0.22 gram of glucose per hour when the blood sugar was 0.196 per cent.; as the blood sugar increased the excretion of sugar decreased, so that when the blood contained 0.24 per cent. the sugar excretion had dropped to 0.17 gram per hour. The rate of excretion increased to 0.216 gram per hour when the blood sugar returned to the original value, 0.196 per cent. In Table VI, Case II, Chart II, Case II, the same observations can be made.

TABLE VI.—CASE II.

J. E. P., March 11, 1916.

Time.		Blood.		Urine.				
A.M.		Hemoglobin. Units.	Sugar. Per cent.	c.c. per hour.	Specific gravity.	Sugar. Per cent.	Sugar. Grams per hour.	Fluid intake. c.c.
9.20	....	65.0	0.20	144	1015	0.5	0.72	
9.30	....	75 grams glucose.	.....	...	....	...	....	450
9.45	¼ hr.	65.0	0.258					
10.05	½ hr.	65.0	0.256					
10.20	¾ hr.	64.0	0.252	52	1013	1.0	0.52	
10.35	1 hr.	64.0	0.329					
11.00	1½ hrs.	63.5	0.368	...	....	...	....	250
11.20								
11.30	2 hrs.	63.5	0.405					
12.45	3¼ hrs.	64.0	0.390	56	1015	1.1	0.61	500
1.30								
3.15	5¾ hrs.	65.0	0.250					
4.15	6¾ hrs.	65.0	0.220	?	1014	0.5	?	
7.35								



Summary. The case reported gave the subjective and objective symptoms of chronic parenchymatous nephritis. Nothing in the patient's history or in the physical finding would lead one to suspect a disorder of carbohydrate metabolism. He had a constant hyperglycemia, but no higher than is ordinarily found in chronic nephritis. The blood sugar curve following the ingestion of glucose was of the type found in non-diabetic nephritis. Of special interest is the fact that glucose constantly appeared in his urine and that the rate of excretion was greatest when the blood sugar concentration was low. A hyperglycemia was accompanied by a decreased excretion of sugar.

Conclusions. Renal diabetes, or better "renal glycuresis,"[22] should be broadly considered as a condition of glycuresis not dependent upon a temporary increase of blood sugar in an individual free from symptoms of diabetes mellitus.

The condition can be recognized by morning synchronous blood and urine sugar tests, the specimens being collected before the patient has had anything to eat or drink.

At least two types can be recognized: (1) Cases with normal or subnormal blood sugar and glycuresis, which is greatly influenced by blood sugar changes; (2) cases with a constant glycuresis which varies with the rate of urinary excretion and is little influenced by blood sugar changes. The possibility of renal glycuresis being in some cases a congenital condition; the question whether or not the clinical course remains permanently free from symptoms of diabetes mellitus; the lack of reserve energy which such a condition produces, if severe; the direct effect upon the kidney of continued excretion of sugar are interesting points to be observed in this condition.

I wish to thank Professor Edward Quintard, director of the department of medicine, and Professor Victor C. Myers, director of the laboratory of pathological chemistry, for helping me in this work.

MULTIPLE ENDOTHELIOMA OF THE SKIN.

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Conception of Endothelioma as an Entity. Dermatological literature records many instances of single and multiple tumors which clinically were thought to be various types of nevi or sarcoma or syringocystadenoma, and which, after microscopic study, were identified as endothelioma by some and as epithelioma or carcinoma by others. Gottheil,[1] for example, published the report of a case of a pigmented tumor of the foot which clinically was considered to be a pigmented sarcoma or melanoma, but on section proved to be an

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[22] Benedict, S. R., and Osterberg, E.: Jour. Biol. Chem., 1918, xxxiv, 258.

[1] Endothelioma of the Skin, Jour .Am. Med. Assn., January 12, 1907, xlviii, p. 93.